Preventing Lead Poisoning in Young Children

A STATEMENT BY THE
CENTERS FOR DISEASE CONTROL
JANUARY 1985

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
CENTERS FOR DISEASE CONTROL

CENTERS FOR DISEASE CONTROL
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CENTER FOR ENVIRONMENTAL HEALTH
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PREFACE

This second revision of the Centers for Disease Control's (CDC's) statement, Preventing Lead Poisoning in Young Children, is more comprehensive than the two previous versions. With help from members of CDC's Ad Hoc Advisory Committee on Childhood Lead Poisoning Prevention and other expert consultants, we have considered new research findings on lead toxicity, redefined lead poisoning at a lower blood lead level, and updated our recommendations on lead-based paint abatement. In addition, a recent article on a new treatment scheme for lead poisoning (symptomatic and asymptomatic) is included.

The precise threshold for the harmful effects of lead on the central nervous system is not known. In the meantime, we have used our best judgment as to what levels of lead are toxic and what practical interventions will lower blood lead levels. As public health officials, our duty is to protect children as best we can--given the limitations of science and the need to make decisions without perfect data. This is the Department of Health and Human Services' major policy statement on the issue.

The progressive removal of lead from leaded gasoline is lowering average blood lead levels in the United States, but the problem of the major source of high blood lead levels in our country--millions of old housing units painted with lead-based paint--is largely unsolved. Until better approaches and more resources are available for removing lead paint hazards in older dwellings where children live, lead poisoning will continue to be a public health problem.

The Committee considered a number of controversial issues, and members vigorously debated until a majority indicated that they could support the point under consideration. Readers should carefully weigh the recommendations in this document, and they should pay particular attention to references to work done since the 1978 CDC statement on lead. This 1984 statement represents agreement of 11 of the 12 Advisory Committee members. One member, Dr. Jerome F. Cole of the International Lead Zinc Research Organization, did not support the recommendations. Minutes of the Advisory Committee meeting on May 17-18, 1984, and Dr. Cole's statement of dissent are available upon request.

ACKNOWLEDGMENTS

The time, effort, and meticulous care the Committee devoted to this statement are gratefully acknowledged. This group of dedicated health professionals, along with notable expert consultants, labored through the results of several years of research in order to gain consensus on extremely complex issues. The various drafts of this document had the benefit of thoughtful suggestions from Committee members and consultants alike. Their work will help protect the children of this nation from this preventable disease for many years to come.

Vernon N. Houk, M.D.
Director
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I. INTRODUCTION

Lead is ubiquitous in the human environment as a result of industrialization. It has no known physiologic value. Excessive absorption of lead is one of the most prevalent and preventable childhood health problems in the United States today. Children are particularly susceptible to its toxic effect.

Since 1970, the detection and management of children exposed to lead has changed substantially. Before the mid-1960's, a level below 60 micrograms of lead per deciliter (ug/dl) of whole blood was not considered dangerous enough to require intervention (Chisolm, 1967). By 1975, the intervention level had declined 50%--to 30 ug/dl (CDC, 1975). In that year, the Center (now Centers) for Disease Control (CDC) published Increased Lead Absorption and Lead Poisoning in Young Children: A Statement by the Center for Disease Control. Since then, new epidemiologic, clinical, and experimental evidence has indicated that lead is toxic at levels previously thought to be nontoxic. Furthermore, it is now generally recognized that lead toxicity is a widespread problem--one that is neither unique to inner city children nor limited to one area of the country.

Progress has been made. The Second National Health and Nutrition Examination Survey (NHANES II) has established average blood lead levels for the U.S. population; lead-contaminated soil and dust have emerged as important contributors to blood lead levels, as has leaded gasoline, through its contribution to soil and dust lead levels. An increasing body of data supports the view that lead, even at levels previously thought to be "safe," is toxic to the developing central nervous system; and screening programs have shown

the extent of lead poisoning in target populations.

A major advance in primary prevention is the phased reduction of lead in gasoline. It is probably responsible for the findings of reduced average blood lead levels in children nationwide (Annest et al., 1983) and in two major cities (Rabinowitz and Needleman, 1982; Billick et al., 1980; Kaul et al., 1983). Lead is no longer allowed in paint to be applied to residential dwellings, furniture, and toys.

The sources of lead are many. They include air, water, and food. Despite the 1977 ruling by the Consumer Product Safety Commission (CPSC) that limits the lead content of newly applied residential paints, millions of housing units still contain previously applied leaded paints. Older houses that are dilapidated or that are being renovated are a particular danger to children. In many urban areas, lead is found in soil (Mielke et al., 1983) and house dust (Charney et al., 1983). Consequently, screening programs--a form of secondary prevention--are still needed to minimize the chance of lead poisoning developing among susceptible young children.

Lead poisoning challenges clinicians, public health authorities, and regulatory agencies to put into action the findings from laboratory and field studies that define the risk for this preventable disease. Although screening programs have been limited, they have reduced the number of children with severe lead-related encephalopathy and other forms of lead poisoning.

The revised recommendations in this 1984 Statement reflect current knowledge concerning screening, diagnosis, treatment, followup, and environmental intervention for children with elevated blood lead levels. Clearly, the goal is to remove lead from the environment of children before it enters their bodies. Until this goal is reached, screening, diagnosis, treatment, followup, and secondary environmental management will continue to be essential public health activities.

DEFINITIONS

The two terms defined below--elevated blood lead level and lead toxicity--are for use in classifying children (whose blood has been tested in screening programs) for followup and treatment. The terms should not be interpreted as implying that a safe level of blood lead has been established. Furthermore, they are to be used as guidelines. They may not be precisely applicable in every case. Each child needs to be evaluated on an individual basis.

The CDC is lowering its definition of an elevated blood lead level from 30 to 25 ug/dl. The definitions below are simplified versions of those in Preventing Lead Poisoning in Young Children: A Statement by the Center for Disease Control: April 1978 (CDC, 1978).

-elevated blood lead level, which reflects excessive absorption of lead, is a confirmed concentration of lead in whole blood of 25 ug/dl or greater;

-lead toxicity is an elevated blood lead level with an erythrocyte protoporphyrin (EP)* level in whole blood of 35 ug/dl or greater.

As defined by blood lead and EP levels, the terms lead toxicity and lead poisoning are used synonymously in this document. "Poisoning" is generally used to describe episodes of acute, obviously symptomatic illness. The term "toxicity" is used more commonly in this document, since screening programs usually involve asymptomatic children.

According to this Statement, the severity of lead toxicity is graded by two distinct scales--one for use in screening, the other for use in clinical management. In the scale used in screening, children with lead toxicity are divided into classes I, II, III, and IV (section IV). These classes indicate the urgency of further diagnostic evaluation (section V). After the diagnostic evaluation, they are placed in one of four risk groups: urgent, high, moderate, and low (section VI).

*EP results are expressed in equivalents of free erythrocyte protopophyrin (FEP) extracted by the ethyl acetate-acetic acid-HCl method and reported in micrograms per deciliter of whole blood. In this Statement, zinc protoporphyrin (ZnPP) and FEP are referred to as EP.

II. BACKGROUND

A nationwide survey, conducted from 1976-1980, showed that children from all geographic areas and socioeconomic groups are at risk of lead poisoning (Mahaffey, Annest et al., 1982). Data from that survey indicate that 3.9% of all U.S. children under the age of 5 years had blood lead levels of 30 ug/dl or more. Extrapolating this to the entire population of children in the United States indicates that an estimated 675,000 children 6 months to 5 years of age had blood lead levels of 30 ug/dl or more. There was, in addition, a marked racial difference in those data. Two percent of white children had elevated blood lead levels, but 12.2% of black children had elevated levels. Further, among black children living in the cores of large cities and in families with annual incomes of less than $6,000, the prevalence of levels of 30 ug/dl or more was 18.6%. Among white children in lower income families, the prevalence of elevated lead levels was eight times that of families with higher incomes.

In the past decade, our knowledge of lead toxicity has greatly increased. Previously, medical attention focused on the effects of severe exposure and resultant high body burdens associated with clinically recognizable signs and symptoms of toxicity (Perlstein and Attala, 1966; Chisolm, 1968; Byers and Lord, 1943). It is now apparent that lower levels of exposure may cause serious behavioral and biochemical changes (De la Burde and Choate, 1972, 1975; NAS, 1976; WHO, 1977). Recent studies have documented lead-associated reductions in the biosynthesis of heme (Piomelli et al., 1982), in concentrations of 1, 25-dihydroxy vitamin D (Rosen et al., 1980; Mahaffey, Rosen et al., 1982), and in the metabolism of erythrocyte pyrimidine (Angle

and McIntire, 1978; Paglia et al., 1977). Results of a growing number of studies indicate that chronic exposure to low levels of lead is associated with altered neurophysiological performance and that the young child is particularly vulnerable to this effect (Needleman et al., 1979; Winneke, 1982; Yule et al., 1981). Investigations have also shown alterations in electroencephalograms (EEG's) (Burchfiel et al., 1980; Benignus et al., 1981; Otto et al., 1982) and decreased velocity in nerve conduction (Seppalainen and Hernberg, 1982; Feldman et al., 1977).

Many factors can affect the absorption, distribution, and toxicity of lead. Children are more exposed to lead than older groups because their normal hand-to-mouth activities introduce many nonfood items into their bodies (Lin-Fu, 1973). Once absorbed, lead is distributed throughout soft tissue and bone. Blood levels reflect the dynamic equilibration between absorption, excretion, and deposition in soft- and hard-tissue compartments (Rabinowitz et al., 1976). Young children absorb and retain more lead on a unit-mass basis than adults. Their bodies also handle lead differently. Higher mineral turnover in bone means that more lead is available to sensitive systems. The child's nutritional status is also significant in determining risks. Deficiencies in iron, calcium, and phosphorus are directly correlated with increased blood lead levels in humans and experimental animals (Mahaffey, 1981; Mahaffey and Michaelson, 1980). Increased dietary fat and decreased dietary intake of calcium (Barltrop and Khoo, 1975; Rosen et al., 1980), iron (Mahaffey-Six and Goyer, 1972), and possibly other nutrients enhance the absorption of lead from the intestine (NAS, 1976; Barltrop and Khoo, 1975).

Since lead accumulates in the body and is only slowly removed, repeated exposures to small amounts over many months may produce elevated blood lead levels.

Lead toxicity is mainly evident in the red blood cells and their precursors, the central and peripheral nervous systems, and the kidneys. Lead also has adverse effects on reproduction in both males and females (Lane, 1949). New data (Needleman et al., 1984) suggest that prenatal exposure to low levels of lead may be related to minor congenital abnormalities. In animals, lead has caused tumors of the kidney. The margin of safety for lead is very small compared with other chemical agents (Royal Commission on Environmental Pollution, 1983).

The heme biosynthetic pathway is one of the biochemical systems most sensitive to lead. An elevated EP level is one of the earliest and most reliable signs of impaired function due to lead. A problem in determining lead levels in blood specimens is that the specimen may be contaminated with lead, and thus the levels obtained may be falsely high. Therefore, in the initial screening of asymptomatic children, the EP level (instead of the lead level) is determined.

The effects of lead toxicity are nonspecific and not readily identifiable. Parents, teachers, and clinicians may identify the altered behaviors as attention disorders, learning disabilities, or emotional disturbances. Because of the large number of children susceptible to lead poisoning, these adverse effects are a major cause for concern.

Symptoms and signs of lead toxicity are fatigue, pallor, malaise, loss of appetite, irritability, sleep disturbance, sudden behavioral change, and developmental regression. More serious symptoms are clumsiness, muscular

irregularities (ataxia), weakness, abdominal pain, persistent vomiting, constipation, and changes in consciousness due to early encephalopathy. Children who display these symptoms urgently need thorough diagnostic evaluations and, should the disease be confirmed, prompt treatment.

In this Statement, screening is distinct from diagnosis. "Screening" means applying detection techniques to large numbers of presumably asymptomatic children to determine if they have been exposed to lead and, if so, what the risks of continued exposure are. Diagnosis, on the other hand, means the categorization of a child appearing to have excess exposure to lead according to the severity of burden and toxicity so that appropriate management can be started. No child with symptoms suggesting lead toxicity should be put through the screening process. He or she should be brought directly to medical attention.

III. SOURCES OF LEAD EXPOSURE

Children may be exposed to lead from a wide variety of man-made sources. All U.S. children are exposed to lead in the air, in dust, and in the normal diet (Figure 1). Airborne lead comes from both mobile and stationary sources. Lead in water can come from piping and distribution systems. Lead in food can come from airborne lead deposited on crops, from contact with "leaded" dust during processing and packaging, and from lead leaching from the seams of lead-soldered cans. In addition to exposure from these sources, some

children, as a result of their typical, normal behavior, can receive high doses of lead through accidental or deliberate mouthing or swallowing of nonfood items. Examples include paint chips, contaminated soil and dust, and, less commonly, solder, curtain weights, bullets, and other items.

LEAD-BASED PAINT

Lead-based paint continues to be the major source of high-dose lead exposure and symptomatic lead poisoning for children in the United States (Chisolm, 1971). Since 1977, household paint must, by regulation, contain no more than 0.06% (600 parts per million (ppm)) lead by dry weight. In the past, some interior paints contained more than 50% (500,000 ppm) lead. The interior surfaces of about 27 million households in this country are contaminated by lead paint produced before the amount of lead in residential paint was controlled. Painted exterior surfaces are also a source of lead. Unfortunately, lead-based paint that is still available for industrial, military, and marine usage occasionally ends up being used in homes.

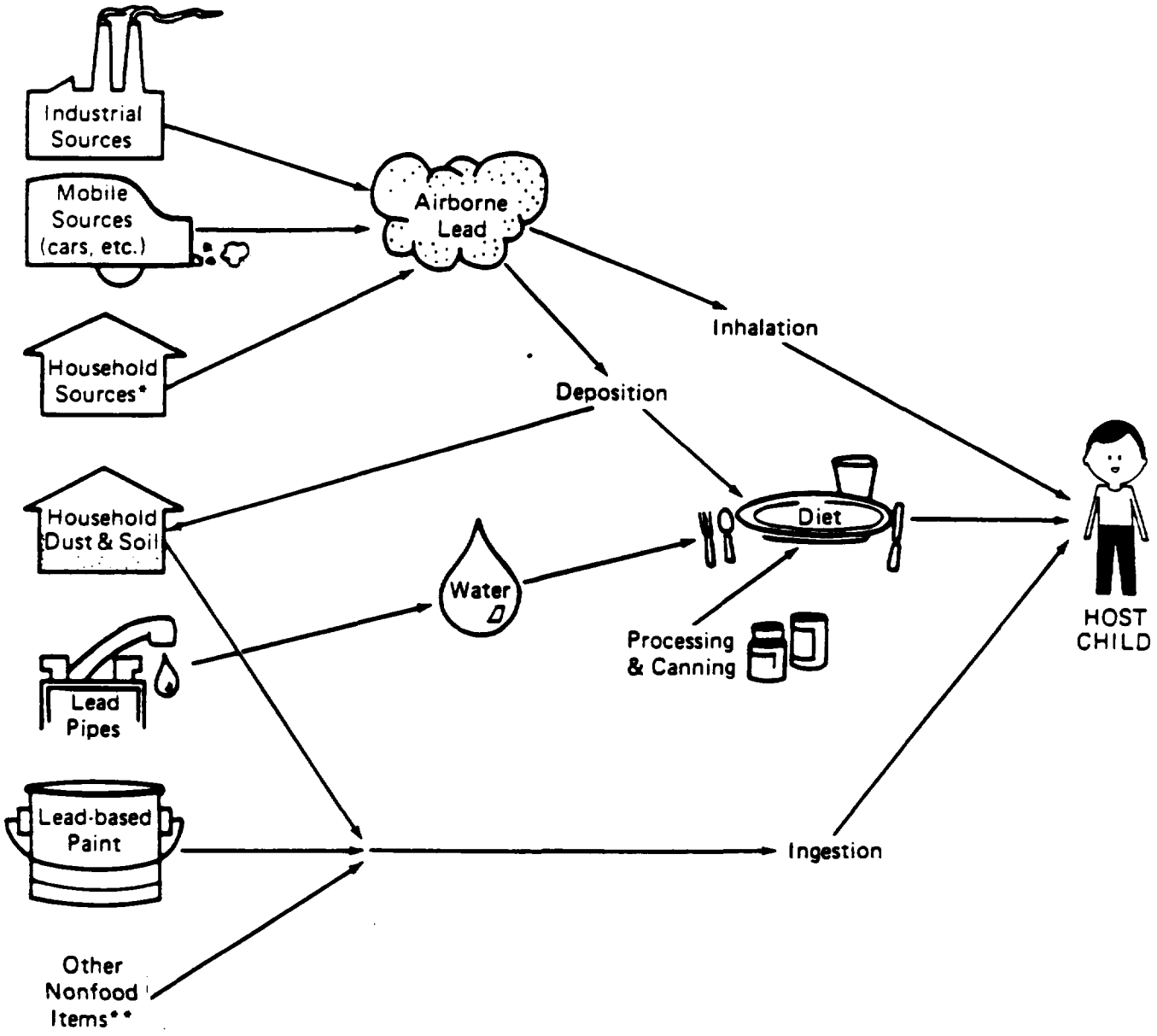
Usually, overt lead poisoning occurs in children under 6 years of age who live in deteriorated housing built before World War II. Pica, the repeated ingestion of nonfood substances, has frequently been implicated in the etiology of lead toxicity in young children. In many cases, however, lead-paint ingestion is simply the result of the normal mouthing behavior of small children who live in lead-contaminated homes. Cases of children poisoned by lead paint have been reported from all regions of the United States and from both urban and rural settings. Increasingly, this poisoning has been reported when families move into a city as "urban homesteaders," and

the children are inadvertently exposed to chips, fumes, or dust from lead-based paint as houses are rehabilitated. Clusters of lead-based paint poisonings have also resulted from demographic shifts within cities, when families with young children have moved into neighborhoods with deteriorating older housing. Increased lead absorption has been reported in children exposed to chips or dust from lead-based paint produced during the deleading of exterior painted steel structures, such as bridges and expressways (Landrigan et al., 1982).

AIRBORNE LEAD

Generally, inhalation of airborne lead is a minor exposure pathway for individual children, but lead-containing particles--airborne and then deposited--can be responsible for high concentrations of lead in dust that children ingest. Studies in New Jersey (Caprio et al., 1974) and California (Johnson et al., 1975) have shown that children living within 100 feet of major roadways have higher blood lead levels than those living farther away. These levels also correlate positively with the average daily traffic volume on roads near homes (Caprio et al., 1974).

Figure 1
SOURCES OF LEAD IN A CHILD'S ENVIRONMENT



*Production of bullets or fishing sinkers
Soldering and stained-glass work
Gasoline sniffing
Pottery glazing
Burning of batteries, colored newsprint, lead-painted objects, and waste oil

**Toys and figures containing lead
Folk remedies
Cosmetics (especially Oriental cosmetics, e.g., Surma, a black eyeliner)
Jewelry (painted with lead to simulate pearl)
Lead-containing dust transmitted on clothing from workplace

Previous estimates of the quantitative relationship between ambient air lead levels and blood lead levels may need to be revised because of new experimental and survey data. Preliminary results from an isotopic lead experiment (Facchetti and Geiss, 1982) suggest that lead from leaded gasoline is a much more important contaminant than it was previously thought to be. The preliminary estimates from that study indicate that at least 25% of the blood lead of residents of Turin, Italy, is derived from lead in gasoline. In Turin, the average blood lead level in adult males is 25 ug/dl; this corresponds to about 6 ug/dl attributable to gasoline.

Data from NHANES II also indicate that leaded gasoline is a more significant source of lead than previously thought. Annest et al. (1983) correlated major reductions in the amounts of lead added to gasoline sold in the United States with significant reductions in children's blood lead levels. They found that between 1976 and 1980, the overall mean blood lead levels in the U.S. population dropped from 14.6 ug/dl to 9.2 ug/dl. A similar relationship between leaded gasoline sales and umbilical cord blood lead levels has been shown by Rabinowitz and Needleman (1983).

Stationary sources can produce concentrated zones of exposure, especially where climatic conditions such as aridity, low wind velocity, and frequent thermal inversions minimize dispersal of airborne lead. The worst situations of this kind in the United States have existed in the vicinity of primary lead smelters (Baker, Hayes et al., 1977).

SOIL AND DUST

Soil and dust that contain lead are often an important source of lead exposure

for children. The particles of airborne lead deposited in soil and dust usually come from automotive, industrial, and similar sources. Flaking lead paint adds to this contamination, particularly in and around houses. In soil, lead tends to remain in the top centimeter, but most soils are contaminated to a much greater depth when the topsoil is disturbed and turned under.

Children appear to obtain lead from dust and soil as a result of their normal exploratory behavior (Barltrop, 1966; Sayre et al., 1974; Roels et al., 1976), coupled in some instances with pica. Because of those mouthing tendencies, young children who live near major sources of airborne lead pollution must be considered at risk of exposure both by inhalation of airborne lead and by ingestion of deposited lead from soil and dust.

In general, lead in soil and dust appears to be responsible for blood lead levels in children increasing above background levels when the concentration in the soil or dust exceeds 500-1,000 ppm.

OCCUPATIONAL SOURCES

Lead dust can cling to the skin, hair, shoes, clothing, and vehicles of workers, and lead can be carried from workplace to home in this way. In a study in Memphis, Tennessee, when a parent worked with lead, the amount of lead in the children's blood correlated with the concentration of lead in dust in their homes (Baker, Folland et al., 1977). Of 91 children tested, 38 (41.8%) had blood lead levels of 30 ug/dl or more, and 10 either had blood lead levels of 80 ug/dl or more or EP levels above 190 ug/dl.

Strict compliance with Occupational Safety and Health Administration (OSHA) standards is quite effective in decreasing this type of exposure. However, many occupational exposures to lead are not covered by the OSHA standards. Companies with fewer than 10 employees (cottage industries, "hobby" production of pottery and stained glasswork, and home manufacturing of bullets and fishing sinkers) are excluded from OSHA standards.

The OSHA standard for lead workers is a blood lead level of 40 ug/dl. In a pregnant woman, lead crosses the placenta, and lead concentrations in umbilical cord blood are nearly equal to those in maternal blood (Barltrop, 1966). Since the growing brain of the fetus is likely to be at least as sensitive to the neurologic effects of lead as the brain of a young child, umbilical cord blood levels should be at least below 25 ug/dl. Therefore, the OSHA standard is probably not sufficiently strict to protect the fetus. Further study is needed to define acceptable lead levels among women of childbearing age.

FOOD AND DRINKING WATER

Lead in food, although rarely responsible for lead poisoning in the United States, is a ubiquitous source of background low-dose exposure for children (Beloian, 1982). Agricultural crops grown near heavily traveled roads or near stationary sources of lead can have significant concentrations because of airborne lead deposited on them. Lead may also be inadvertently added to foods during processing and handling. Canned foods may have particularly high lead contents, because acidic foods can leach lead from the solder in the seams of the cans (Lamm et al., 1973).

Generally, lead in drinking water has been leached from pipes and soldered joints by soft water having an acidic pH. Severe lead exposure has been reported among children in Glasgow, Scotland, where pure, acidic water was allowed to stand overnight in attic cisterns lined with lead (Beattie et al., 1972). The problem was alleviated by changing the pH of the water in the water treatment plant. In the United States, lead water pipes are most commonly found in older sections of northeastern cities and, occasionally, in rural areas of the northeast (Morse et al., 1979).

LEAD-GLAZED POTTERY

Although not a widespread source of lead, lead-glazed pottery can release large amounts of lead into food and drink. It has been responsible for outbreaks of serious poisoning (Klein et al., 1970). In several episodes reported to CDC, the pottery had been imported. Homemade or craft pottery and porcelain-glazed vessels have been found to release large quantities of lead, particularly if the glaze is chipped, cracked, or improperly applied (Osterud et al., 1973). If the vessels are repeatedly washed, the glaze may deteriorate and pottery previously tested as safe can become unsafe (D. M. Wallace, personal communication).

OTHER SOURCES

Lead is found in a variety of items, some of which endanger specific populations or ethnic groups. A variety of folk remedies contain lead, including azarcon and greta used by Mexican groups and pay-loo-ah used by Hmong refugees from Laos. Serious poisoning can also result from gasoline sniffing; the burning of waste oil, colored newsprint, battery casings, or lead-painted wood; and target practice in poorly ventilated, indoor firing ranges.

IV. SCREENING

GOAL OF A CHILDHOOD LEAD POISONING PREVENTION PROGRAM

The goal of a childhood lead poisoning screening program is to identify children with significant exposure to lead early enough to prevent serious toxicity. Elevated blood lead levels must be detected in asymptomatic children, and appropriate medical and environmental interventions must follow. The goal can be reached only through--

1. A screening program that enrolls the maximum number of children in high-risk populations.

2. A referral system that ensures a comprehensive diagnostic evaluation of every child with a positive screening test.

3. A program that assures identification and elimination of the

source(s) of the child's lead exposure.

4. A system that monitors the adequacy of the treatment and the followup of each child with a diagnosis of lead toxicity.

Screening is of no value without prompt, thorough, and continuing medical and environmental followup for those children found to have lead toxicity--that is, as stated earlier, an elevated blood lead level (a confirmed concentration in whole blood of 25 ug/dl or greater) and an EP level in whole blood of 35 ug/dl or greater. Also as stated earlier, screening must be distinguished from diagnosis:

Screening refers to the testing of large numbers of children considered to be ASYMPTOMATIC in order to identify those who need further evaluation. Diagnosis, on the other hand, refers to categorizing a child's condition according to severity of lead burden and toxicity. Then, on the basis of the category, management is selected.

Children whose elevated blood lead levels are detected by screening should be brought directly to medical attention, and the diagnostic process should be started without delay. Children with symptoms suggestive of lead poisoning require urgent and thorough diagnostic evaluation and, if the diagnosis is confirmed, immediate treatment. The symptoms of lead poisoning are nonspecific; they are described in section V.

TARGET POPULATION

Lead is most harmful to children between the ages of 9 months and 6 years. Ideally, all children in this age group should be screened. As more children are screened for iron deficiency with EP testing, simultaneous lead screening of these same groups becomes feasible. The list of priority groups in Table 1 highlights groups for which screening is strongly indicated. Testing children in low-risk groups for lead toxicity may not be practical unless it is done simultaneously with EP tests for iron deficiency.

Children in the 12- to 36-month-old age group who live in or are frequent visitors in deteriorating older buildings (including day-care centers) make up the highest priority group.

Siblings, housemates, and playmates of children with identified lead toxicity probably have similar exposures to lead, and they should be promptly screened. Suggested rankings for these and other priority groups are in Table 2.

Table 1

Suggested Priority Groups for Lead Screening

Priority	
1.	HIGHEST--Children, age 12 to 36 months, who live in or are frequent visitors in older, dilapidated housing
2.	Children, age 9 months to 6 years, who are siblings,

housemates, visitors, and playmates of children with known lead toxicity

3. Children, age 9 months to 6 years, living in older, dilapidated housing

4. Children, age 9 months to 6 years, who live near lead smelters and processing plants or whose parents or other household members participate in a lead-related occupation or hobby

5. Children, age 9 months to 6 years, who live near highways with heavy traffic or near hazardous waste sites where lead is a major pollutant

6. All children 12 to 36 months of age

7. All children 9 months to 6 years of age

SCREENING SCHEDULE

Screening for lead poisoning should be incorporated into a general pediatric health care program, and children in the target population should be screened at least once a year. The first screening should be done when the child is between 9 and 12 months old. Children generally have higher blood lead levels between May and October (NAS, 1976), so screening efforts should be concentrated in those months. Since negative screening tests in children living in a hazardous environment do not rule out subsequent exposure, children 12 to 36 months old who are at high risk of exposure should be screened every 2 to 3 months, especially during the summer. Children who move into a high-risk area after age 3 years may also need to be screened more than once a year.

SCREENING METHODS

Currently, the most useful screening tests are those for erythrocyte protoporphyrin (EP) and blood lead. Venous or capillary blood can be used for both tests, but capillary specimens are easier to collect and are, therefore, more widely used. Capillary blood may be transported in a capillary tube with an anticoagulant or dried on filter paper. Sampling methods used in the field must be compatible with laboratory capabilities.

EP and blood lead tests measure different aspects of lead toxicity. As stated earlier, EP tests measure the level of EP in whole blood, and a level of 35 ug/dl or more indicates impaired heme synthesis, which may be due to the toxic effects of lead; blood lead tests measure lead absorption, and a confirmed

concentration of 25 ug/dl or more, referred to as an elevated blood lead level, reflects an excessive absorption of lead. Usually, there is a close correlation between results of the two tests for specimens from the same child, but, occasionally, the result of one test may be elevated and the result of the other, not elevated. The EP test has three advantages over the blood lead test: (1) when blood lead levels are moderately elevated, the EP test better identifies children with rising blood lead levels (Reigart and Whitlock, 1976); (2) if the specimen is contaminated with lead, the contamination does not affect the EP test; and (3) the EP test is an accepted screening test for iron deficiency.

INTERVENTION LEVELS

Children screened for lead poisoning can be grouped into two categories: those who require further evaluation and those who do not. Choosing the intervention level that divides these two groups is based on a compromise among the following:

(1) the desire to identify all children with any degree of lead toxicity
(2) a judgment about the urgency of preventing various detectable effects
(3) the sensitivity and specificity of a practical screening test
(4) society's ability to remove the sources of lead exposure

A. Pathophysiological Considerations

In recent years, levels of exposure previously considered "safe" have been shown to produce adverse effects. In addition, contemporary people (including children) living in remote areas with negligible exposure to lead have blood lead levels much lower than people living in the United States (Piomelli, 1980). Thus, the blood lead levels of U.S. children reflect a high degree of environmental contamination by lead. Today, the average blood lead level in the U.S. population is about 10 ug/dl--approximately three times the average level found in some remote populations. These observations suggest that the average level in the U.S.A. should be reduced. At present, however, because of practical considerations, the goal of reducing U.S. levels to those of remote populations is unattainable. Therefore, the blood lead level at which intervening action should be taken should be based on (1) criteria that indicate significant risk to the individual child and (2) the best combination of tests: a test for the blood lead level as an indicator of absorption and a test for EP as an indicator of biochemical derangement.

Since the CDC's 1978 statement on lead poisoning, several investigators have demonstrated effects of low-level lead exposures in these areas:

1. children's behavior and intelligence (Needleman et al., 1979; Winneke, 1982; Yule et al., 1981)
2. the central and peripheral nervous systems of adult workers (Mantere et al., 1982; Seppalainen and Hernberg, 1982)
3. heme biosynthesis in children (Piomelli et al., 1982)
4. nucleotide metabolism (Angle and McIntire, 1978)

venous specimens collected from children throughout New York City. Piomelli and his colleagues were trying to find the blood lead level at which the EP level began to increase. A variety of statistical techniques were used, and the findings were consistent; when blood lead levels increased linearly above the area of 15-18 ug/dl, the EP level increased exponentially.

Recent studies of EDTA (calcium disodium ethylene diamine tetraacetic acid) mobilization testing indicate that the amount of lead excreted by children with blood lead levels of 30-40 ug/dl may often be comparable to that excreted by children with levels of 50-70 ug/dl (Markowitz and Rosen, 1984). This finding suggests that the blood lead level may underestimate the body burden of lead.

In summary, the EP data, with data from the other studies referred to, indicate that the 1978 blood lead guideline of 30 ug/dl has little or no margin of safety and should be lowered.

B. Practical Considerations

Although the biologic threshold for lead toxicity, as manifested by increasing EP levels, is less than 20 ug/dl, the criteria for a screening program have to take into account additional factors: (1) acceptability, sensitivity, and specificity of the screening procedure; (2) cost-effectiveness; and (3) the feasibility of effective intervention and followup.

The identification of children with blood leads below 25 ug/dl would require screening with a blood lead assay rather than an EP test, since the latter

screening test has a very poor sensitivity and specificity below a blood lead level of 25 ug/dl. Such a recommendation would require most programs to use venous blood samples or to adopt impractically rigorous training and quality control procedures. Capillary blood samples are prone to environmental contamination with lead. In most programs, particularly those in high-risk areas, the use of "routine" capillary blood-drawing techniques results in an unacceptably high frequency of falsely elevated blood lead levels. On the other hand, taking blood samples from the veins of small children is less acceptable to parents and technically much more difficult. In addition, the cost of the screening program would increase severalfold. For all these reasons, an intervention level set at blood lead values below 25 ug/dl might ultimately substantially decrease the number of children being screened.

If local screening programs evaluate the distribution of moderately and highly elevated blood lead levels within a community, the findings may identify sources of lead that might go unnoticed if the program focused solely on children with high lead levels.

Even when slightly elevated blood lead levels are found, some interventions are possible and effective. House dust is an insidious but apparently effective carrier of lead to children in contaminated environments. In urban areas dust frequently contains large amounts of lead, thought to come primarily from airborne sources or leaded paint. Charney et al. (1983) have documented the effectiveness of controlling house dust; thus, in some situations, low and moderately elevated blood lead levels can be reduced simply by controlling house dust.

Considering these factors, CDC recommends as the intervention level a blood lead level of 25 ug/dl associated with an EP level of 35 ug/dl. When the blood lead level is 25 ug/dl or greater associated with an EP value of 35 ug/dl or greater, lead toxicity is present; identification of such children is the focus of CDC's new recommendations. Children may have mildly elevated blood lead levels without concurrent increases in EP concentrations, and it is desirable to identify these children, but, at present, this is impractical and beyond the criteria set for screening programs. Nonetheless, when resources and capabilities permit both blood lead and EP to be measured in the primary screening program, additional children with elevated blood lead levels will be identified. A more practical method for identifying such children needs to be developed.

The CDC recommends the following cutoff levels for determining a high risk for lead toxicity: for EP screening, a level of 35 ug/dl (whole blood should be tested); for followup testing, all children with a blood lead of 25 ug/dl or more should be considered at risk for the toxic effects of lead. Since the EP level is also elevated in iron deficiency, an elevated EP test alone should not be considered to be diagnostic of lead toxicity.

MEASUREMENT OF ERYTHROCYTE PROTOPORPHYRIN

Erythrocyte protoporphyrin (EP) may be measured by fluorometry after it has been extracted from the red blood cells or by direct fluorescence in intact cells (Lamola et al., 1975; Blumberg et al., 1977). In lead toxicity and iron deficiency, this metabolite is present in the red cells mainly as zinc

protoporphyrin (ZnPP), but the ethyl acetate-acetic acid extraction procedure converts zinc protoporphyrin to erythrocyte protoporphyrin. Measuring ZnPP by hematofluorometer and EP after it has been extracted from the cells reflects essentially the same compound. In erythropoietic protoporphyria, an extremely rare disease, EP is markedly elevated--usually above 300 ug/dl. This is the free EP base, but it is detected by the EP extraction method and, to a lesser extent, by the hematofluorometer.

EP is also elevated in sickle cell anemia and other hemolytic anemias (Langer et al., 1972). Hyperbilirubinemia (jaundice) will cause falsely elevated EP readings with the hematofluorometer, but not with the extraction method (Buhrmann et al., 1978). Recent colds, ear infections, and other minor illnesses may also cause slight elevations of EP (Reeves et al., 1984). Nevertheless, lead toxicity should always be ruled out as the cause of elevated EP levels.

A. Use of Hematofluorometers

Hematofluorometers measure ZnPP and report values in EP equivalents. They are calibrated against the extraction method and theoretically should yield corresponding values. In practice, the values obtained with these instruments are usually not 100% of the EP present and may be considerably less. At least two studies (Kaul et al., 1983; Hammond et al., 1984) indicate that, at high

levels, values obtained with hematofluorometers are lower than those obtained with the extraction method, but that up to 35 ug/dl the results are similar. Because hematofluorometers give immediate results and are economical, they are eminently suitable for field screening.

For both the hematofluorometer and the extraction method, the distinction between a positive and negative screening test should be based on a cutoff level of 35 ug/dl. However, for risk classification, the cutoff points for ZnPP measured by hematofluorometer (Table 2.A) differ from those for EP measured by the extraction method (Table 2.B). If possible, centralized laboratories should use extraction methods, and, if the followup laboratory has extraction capability, all confirmatory tests for EP should be done by extraction, not hematofluorometer. Hematofluorometers are most likely to give accurate results when used to analyze freshly collected blood specimens. The differences between methods need further study.

B. Erythrocyte Protoporphyrin and Iron Deficiency

A benefit of EP screening is that when an elevated EP level proves not to be due to lead, it usually reflects iron deficiency (Piomelli, 1977). The first signs of iron deficiency are biochemical abnormalities (low serum ferritin, low transferrin saturation, and high EP) followed by cellular abnormalities (microcytosis and hypochromia). Iron deficiency anemia follows these changes as the hemoglobin and hematocrit values fall.

The EP test proved to be practical in screening for iron deficiency in a population of 4,160 children (Yip et al., 1983). The upper limit of normal

for EP in this study was 35 ug/dl. The predictive value appeared to be satisfactory.

Iron deficiency is common in many of the groups at risk for lead poisoning--especially among inner-city children of low socioeconomic status living in old, dilapidated housing. Iron deficiency is common among infants ages 9 to 24 months; the highest frequency of lead poisoning extends through 36 months. Iron deficiency and lead toxicity may occur in the same child. Furthermore, experimental evidence indicates that iron deficiency increases the proportion of lead absorbed from the intestine and aggravates the toxic effects of lead.

Analysis of the NHANES II data has clarified the relationship between elevated EP values, blood lead levels, and iron deficiency in a representative sample of the U.S. population. Among children in the NHANES II survey with elevated EP values, 31% have elevated blood lead levels, 18% have iron deficiency (as evidenced by a transferrin saturation of less than or equal to 12%), and 11% have evidence of both conditions (R. Yip, personal communication). On the other hand, among children with elevated blood lead levels, only about 26% have lead toxicity--that is, an elevated EP level (NCHS, 1984). In high priority populations (Table 1), in which iron deficiency is more common and lead levels are higher, a greater proportion of children with elevated blood lead levels would have elevated EP levels. Analyses by both Yip and NCHS confirm that a synergistic effect exists between lead toxicity and iron deficiency in children, as experimental studies in animals have suggested.

MEASUREMENT OF BLOOD LEAD

Unlike elevated EP levels (which may be caused by iron deficiency or other illnesses), elevated blood lead levels are specific for lead absorption. Fluctuations in blood lead values over a short period can be due to physiologic variations or sporadic acute lead exposure.

Capillary samples are highly sensitive to contamination with environmental lead. If such samples are to be taken for blood lead assays, the personnel must be rigorously trained before any screening program is begun, and duplicate capillary blood specimens should be drawn. A single tube of capillary blood should never be used for the diagnosis of elevated blood lead, because an elevated value may be caused by contamination. If results of tests for blood lead from two tubes differ substantially, the higher value can be considered spurious. Even when the results are equally elevated, contamination cannot be excluded. Therefore, only venous blood samples should be used to confirm a diagnosis or to determine or assess treatment. There is less likelihood of contamination in a venipuncture, but venous blood may be difficult to collect from very young children. Neither the blood lead nor the extraction EP test should be considered a routine procedure in the clinical laboratory. To help insure credible test results, laboratories performing these tests should participate in the CDC proficiency testing program or the equivalent.

SCREENING SCHEMES

Three feasible screening strategies are--

1. Screening with EP tests, followed by blood lead measurements if indicated. This is the most common procedure.

2. Screening with both EP and blood lead tests.

3. Screening with blood lead tests, followed by EP measurements if indicated.

The CDC recommends EP tests, followed by blood lead measurements for all children with an elevated EP level. The EP test has these advantages:

1. Ease of measurement by hematofluorometer or the extraction method.

2. Results that are not affected if specimens are contaminated with environmental lead.

3. More cost effective than screening with the blood lead test.

4. Ability to detect a child's metabolic response to the toxicity of lead.

5. Possibility of differentiating between children with stable blood lead levels and those with declining levels.

6. Possibility of identifying children who have iron deficiency.

In some areas, where the environment is grossly contaminated with lead, a strategy of simultaneous testing for EP and blood lead levels is recommended. In these cases, venous samples should be used for measuring lead.

When EP is the primary screening tool, two approaches are possible:

1. EP measured on site. A capillary blood specimen is collected, and while the child waits at the screening site, EP is determined by hematofluorometer. Children found to have EP values of 34 ug/dl or less are discharged until the next routine screening.

   For those with EP values of 35 ug/dl or more, additional blood samples are taken (preferably by venipuncture) for laboratory analysis of blood lead and of EP--by extraction, if the method is available.

2. EP measured off site. A venous blood sample or duplicate capillary samples are collected at the screening site and sent to the laboratory for measurement of EP, preferably by the extraction method. The amount of blood collected should be sufficient for confirmatory tests. Unused specimens of blood from children whose EP levels are 34 ug/dl or less may be discarded. For those children with EP levels of 35 ug/dl or more, the blood lead levels and hematocrits or hemoglobin concentrations should be determined.

On site, EP is nearly always measured by hematofluorometer; off site--preferably--it is measured by the extraction method. If the blood

specimen is protected from temperature extremes and light, it may be stored for a week to 10 days before being analyzed by the extraction method. Blood collected on filter paper may be stored for several weeks before it is analyzed.

INTERPRETATION OF SCREENING RESULTS

A single screening test, either for EP or blood lead, cannot be used to categorize children for priority in followup. Both EP and blood lead levels must be used to determine the potential risk of lead toxicity in the children screened.

Children can be arbitrarily divided into four classes on the basis of EP and blood lead screening results. In view of the observed discrepancy between results from the hematofluorometer and extraction methods, two tables are given: Tables 2.A and 2.B (derived from Kaul et al., 1983). This classification merely suggests the relative risk and the priority for medical evaluation and environmental intervention, and the tables should be used only as general guidelines. Children 12 to 36 months old should be given priority over older ones, and children whose EP and blood lead levels fall into the upper range of a class should be given priority over those whose levels fall into the lower range. For example, the urgency for followup is greater for a 1-year-old whose EP level by extraction is 109 ug/dl and whose blood lead level is 49 ug/dl than for a 5 1/2-year-old whose EP level, by the extraction method, is 36 ug/dl and whose blood lead level is 26 ug/dl. Yet both children fall into class II.

Children in class IV--at urgent risk of lead toxicity--should be medically evaluated within 24 hours, and in no case later than within 48 hours. Children in class III are at high risk. Those in class II are at moderate risk, and those in class I, at low risk.

Class I can be subdivided into two additional categories. Class Ia (blood lead 25 ug/dl or less and EP, 35 ug/dl or more) includes children with iron deficiency. These children should be retested, with additional assessment of iron status. Class Ib (blood lead, 25-40 ug/dl, and EP, less than 35 ug/dl) covers children who appear to have transient, stable, declining, or increasing blood lead levels. Results should be confirmed by retesting, and the children should be carefully followed. In some cases, the blood lead and EP results will differ. When the EP value is significantly higher than the value suggested by the blood lead level, the child probably has both iron deficiency and excessive lead absorption.

Screening should focus on asymptomatic children. Children with symptoms should be referred for immediate evaluation, regardless of their risk classification.

TABLE 2.A - ZINC PROTOPORPHYRIN (ZnPP) BY HEMATOFLUOROMETER

RISK CLASSIFICATION OF ASYMPTOMATIC CHILDREN FOR PRIORITY MEDICAL EVALUATION

Blood Lead #	Erythrocyte Protoporphyrin (EP) #			
	<35	35-74	75-174	>175
Not done	I	*	*	*
<24	I	Ia	Ia	EPP+
25-49	Ib	II	III	III
50-69	**	III	III	IV
>70	**	**	IV	IV

\# = Units are in ug/dl of whole blood.

* = Blood lead test needed to estimate risk.

EPP+ = Erythropoietic protoporphyria. Iron deficiency may cause elevated EP levels up to 300 ug/dl, but this is rare.

** = In practice, this combination of results is not generally observed; if it is observed, immediately retest with whole blood.

NOTE: Diagnostic evaluation is more urgent than the classification indicates

for--

1. Children with any symptoms compatible with lead toxicity.

2. Children under 36 months of age.

3. Children whose blood lead and EP levels place them in the upper part of a particular class.

4. Children whose siblings are in a higher class.

These guidelines refer to the interpretation of screening results, but the final diagnosis and disposition rest on a more complete medical and laboratory examination of the child.

TABLE 2.B - ERYTHROCYTE PROTOPORPHYRIN (EP) BY EXTRACTION

RISK CLASSIFICATION OF ASYMPTOMATIC CHILDREN FOR PRIORITY MEDICAL EVALUATION

	Erythrocyte Protoporphyrin (EP) #			
Blood Lead #	<35	35-109	110-249	>250
Not done	I	*	*	*
<24	I	Ia	Ia	EPP+
25-49	Ib	II	III	III
50-69	**	III	III	IV
>70	**	**	IV	IV

\# = Units are in ug/dl of whole blood.

* = Blood lead test needed to estimate risk.

EPP+ = Erythropoietic protoporphyria. Iron deficiency may cause elevated EP levels up to 300 ug/dl, but this is rare.

** = In practice, this combination of results is not generally observed; if it is observed, immediately retest with venous blood.

NOTE: Diagnostic evaluation is more urgent than the classification indicates for--

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1. Children with any symptoms compatible with lead toxicity.

2. Children under 36 months of age.

3. Children whose blood lead and EP levels place them in the upper part of a particular class.

4. Children whose siblings are in a higher class.

These guidelines refer to the interpretation of screening results, but the final diagnosis and disposition rest on a more complete medical and laboratory examination of the child.

V. DIAGNOSTIC EVALUATION

Screening tests are not diagnostic. Therefore, every child with a positive screening test should be referred to a physician for evaluation, with the degree of urgency indicated by the risk classification. At the first diagnostic evaluation, if the screening test was done on capillary blood, a venous blood lead level should be determined in a laboratory that participates in CDC's blood lead proficiency testing program. Even when tests are done by experienced personnel, blood lead levels may vary 10% to 15%, depending on the level being tested. Tests for the same child may vary as much as ±5 ug/dl in a 24-hour period. Thus, differences of 1 to 5 ug/dl between screening and diagnostic levels in either direction should not necessarily be interpreted as indicative of actual changes in the child's lead absorption or excretion.

Additional blood samples may be needed for tests such as complete blood counts, serum iron, total iron binding capacity, and serum ferritin. The amounts necessary for these tests, which usually exceed the amount obtainable by capillary sample, can be obtained with a single venipuncture.

Symptoms, if present, constitute an urgent risk, warranting prompt hospitalization (see section VI). Symptoms must be looked for, and they can be missed (Piomelli et al., 1984):

> Acute lead encephalopathy is characterized clinically by some or all of these symptoms: coma, seizures, bizarre behavior, ataxia, apathy, incoordination, vomiting, alteration in the state of consciousness, and subtle loss of recently acquired skills. Any one or a mixture of these

symptoms, associated with an elevated blood lead level, constitutes an acute medical emergency. Lead encephalopathy is almost always associated with a blood lead level exceeding 100 ug/dl, although, occasionally, it has been reported at blood lead levels as low as 70 ug/dl.

Symptomatic lead poisoning without encephalopathy is characterized by one or several symptoms: decrease in play activity, lethargy, anorexia, sporadic vomiting, intermittent abdominal pain, and constipation. It is usually associated with a blood lead level above 70 ug/dl, although, occasionally, cases are associated with a level as low as 50 ug/dl. <u>If the blood lead level is below 50 ug/dl, other causes should be vigorously sought</u>. Since any symptomatic child may develop acute lead encephalopathy, treatment and supportive measures must be started immediately on an emergency basis.

Whether or not symptomatic lead poisoning is present, the child should have a complete pediatric evaluation. Special attention should be given to--

1. A detailed history, including the presence or absence of clinical symptoms, child's mouthing activities, existence of pica, nutritional status, family history of lead poisoning, possible source of exposure, and previous blood lead or EP determinations.

2. The physical examination, especially the neurologic examination.

3. Nutritional status and hematologic evaluation for iron deficiency. Iron deficiency contributes to an elevated EP and can enhance lead absorption and toxicity.

4. Confirmatory diagnostic tests.

5. Trends in EP and blood lead levels.

Since trends are important in diagnosis and management, serial measurements of blood lead and EP (and other measurements as indicated) are far more valuable than data obtained at one time. To be comparable and interpretable, serial EP levels should be analyzed by the same method.

Probably the most reliable method for determining the source of exposure is obtaining a careful, complete environmental history (see section III), inspecting the home for lead hazards, and learning about the child's hand-to-mouth behavior through careful questioning. Pica, the Latin word for "magpie," describes the habitual ingestion of nonfood substances. This should not be regarded as synonymous with the normal oral behaviors of small children, such as finger and thumb sucking and nail biting.

An initial plan for management requires that all interacting factors be taken into account. The plan should be modified as indicated by long-term trends in lead absorption, exposure, and clinical status.

TESTS

In addition to confirmatory and serial EP and blood lead determinations, the following tests can be useful (if available) in assessing the patient's lead absorption status.

1. Tests for Iron Deficiency

Because the EP can reflect iron deficiency as well as lead exposure, the presence of iron deficiency must be established or ruled out if EP levels are to be properly interpreted.

A common misconception is that a child with a "normal" hematocrit (33% or more) or hemoglobin concentration (11 g/dl or more) could not be iron deficient. This is not true, particularly with respect to iron deficiency sufficient to affect EP and, worse, to enhance lead absorption and retention. Thus, although a complete blood count (CBC) and a reticulocyte count are indicated in the evaluation of lead toxicity, they are not sensitive enough to rule out iron deficiency.

Of the red blood cell (RBC) indices, a decreased mean corpuscular volume (MCV) is a useful indicator of iron deficiency. Normal values depend on age (Dallman, 1982).

Serum iron and iron binding capacity are more sensitive than the MCV. In general, an elevated iron binding capacity of more than 350 ug/dl is more likely to accurately indicate iron deficiency than a normal or low serum iron, since the serum iron is quite sensitive to both dietary iron and diurnal variation. Thus, if a child has eaten an iron-rich food within 2-4 hours before the blood for the test is drawn, the result may be closer to the normal level than is actually the case. Under standardized conditions, an abnormally low ratio of serum iron to iron binding capacity (transferrin saturation) is consistent with iron deficiency. In addition to the level of EP itself, the serum ferritin level is an accurate indication of overall iron status.

2. Flat Plate of the Abdomen

Radiologic examination (flat plate) of the abdomen may reveal radiopaque foreign material, but only if the material has been ingested during the preceding 24 to 36 hours. Since lead ingestion is sporadic, this examination is significant only if the results are positive; negative results do not rule out lead poisoning. Positive results indicate recent ingestion of large amounts of lead.

3. X-ray of Long Bones

X-rays of the long bones, usually the knees, may help estimate the duration of exposure. Lines of increased density in the metaphyseal plate of the distal femur and proximal tibia and fibula are "growth arrest lines." They are caused by lead, which disrupts the metabolism of the bone matrix. As a result, areas of increased mineralization or calcification may be present at

the metaphyses of the long bones. Though sometimes called "lead lines," they are not an x-ray shadow of deposited lead.

Although definitive data are not available, these lines are thought to become visible after at least 4 to 8 weeks from the time exposure began; the length of time depends on the age of the child and the degree of lead exposure. The width and intensity of the lines reflect prolonged previous lead absorption but do not indicate current ingestion. They are seldom seen in children under 24 months of age. Negative x-rays do not rule out lead poisoning.

4. Calcium Disodium EDTA Mobilization (or Provocative Test)

This test is used to identify children who will respond to chelation therapy with a brisk lead diuresis. Children whose blood lead level exceeds 55 ug/dl should not receive a provocative chelation test. Instead, appropriate chelation therapy should be started. The mobilization test is particularly useful when the screening test indicates that the child has lead toxicity and there is some question as to whether chelation therapy is indicated. This test provides an index of the mobile or potentially toxic fraction of the total body lead burden (Saenger et al., 1982).

Since CDC's 1978 statement, an 8-hour mobilization test has been shown to be as reliable as a 24-hour mobilization test (Markowitz and Rosen, 1984). Although an 8-hour test may be done on an outpatient basis, the patient should not leave the clinic. The careful use of "lead-free" apparatus is mandatory.*

* Special lead-free collection apparatus must be used if valid test results are to be obtained. The laboratory performing the analysis may supply the proper collection apparatus. Preferably, urine should be voided directly into polyethylene or polypropylene bottles that have been cleaned by the usual procedures, then washed in 1% nitric acid, and thoroughly rinsed with deionized, distilled water. For children who are not toilet trained, plastic pediatric urine collectors, with double compartments, may be used. Urine collected in this manner should be transferred directly to the urine collection bottles. Preserving the collected urine with hydrochloric acid will stabilize not only lead but also d-aminolevulinic acid (ALA).

5. Lumbar Puncture

CAUTION:

If a lumbar puncture is needed to rule out meningitis or other serious disease, it should be performed cautiously and only after a careful search for signs and symptoms of increased intracranial pressure. The fluid should be obtained drop by drop, and no more than 1 milliliter (ml) should be removed.

The following tests are not useful in diagnosing lead toxicity.

1. Microscopic Examination of Red Cells for Basophilic Stippling

Since basophilic stippling is not universally found in chronic clinical lead poisoning and is relatively insensitive to lesser degrees of lead toxicity, it is not considered useful in diagnosis.

2. Tests of Hair and Fingernails for Lead Levels

The levels of lead in hair or fingernails are not well correlated with blood lead levels; therefore, tests for these levels are not considered useful in diagnosis.

VI. CLINICAL MANAGEMENT

The system described in section IV is for an initial classification, to be modified by results of the diagnostic evaluation. Thus, after all information is available to the clinician, the child's true risk classification is established. Clinical management includes eliminating the source of the child's lead exposure; providing general pediatric care, family education, and, when appropriate, chelation therapy; and correcting any nutritional deficiencies. In addition, followup examinations must be performed until the risk of further damage is minimal. The single most important factor in pediatric management is to reduce the amount of lead ingested. The family must be fully informed of the child's condition and of the clinical and environmental actions to follow.

One recommended approach to the treatment of children with symptomatic and asymptomatic lead poisoning is described in detail in the Appendix. The major new feature of this approach is an increased reliance on calcium disodium EDTA mobilization testing among children with moderate blood lead levels. The test results are used to decide whether chelation is indicated. A full course of chelation therapy should not be given without either a confirmed blood lead level equal to or greater than 56 ug/dl or a positive mobilization test in children with blood lead levels of 25-55 ug/dl. This approach is recommended by four major medical centers in which the staffs have had extensive experience in the diagnosis and treatment of children with lead poisoning.

The cornerstones of clinical management are careful clinical and laboratory surveillance of the child and a reduction in lead exposure to prevent further accumulation of lead. This approach allows previously absorbed lead to be slowly excreted. Most children with lead toxicity do not require chelation therapy, but those who do may need more than one course of treatment.

The followup program for asymptomatic children depends upon the degree of risk determined during the diagnostic evaluation.

For the purposes of clinical management and followup, the risk categories are ranked from urgent to low.

Urgent - Blood lead levels of 70 ug/dl or more with or without symptoms.

High - Children whose repeat EP and confirmatory venous blood lead levels fall in the class II and III ranges of the screening test, but who also

have a positive calcium disodium EDTA mobilization test or other confirmatory diagnostic tests or risk factors. Children in class III who have not had confirmatory diagnostic tests should be considered high risk until evidence places them in another risk category.

Moderate - Children whose repeat EP and venous blood lead levels fall into the class II range of the screening test but whose other confirmatory diagnostic tests are negative.

Low - Children whose repeat EP and venous blood lead levels fall into the class I range of the screening tests. These children are usually not given other diagnostic tests.

This categorization is arbitrary and can be adapted to a particular child. For example, a 20-month old with persistent pica whose environmental lead hazard cannot be controlled satisfactorily may be considered high risk, even if his or her repeat EP and venous blood lead levels fall in the range of class II and other diagnostic tests are negative.

URGENT RISK

Children with blood lead levels of 70 ug/dl or more, regardless of the presence or absence of clinical symptoms, should be treated with the same intensity as children with frank neurologic manifestations. The higher the confirmed venous blood lead, the greater the need for chelation therapy. Severe and permanent brain damage may occur in as many as 80% of children who have acute encephalopathy (Perlstein and Attala, 1966). Treatment before onset of encephalopathy will improve this grim prognosis.

Lead toxicity is a chronic medical problem. Children who require chelation therapy will need long-term medical surveillance and care. The EP levels can fluctuate during and immediately after chelation therapy. After an apparently successful course of therapy with calcium disodium EDTA (incorporating BAL, British Anti-Lewisite, as necessary), the "rebound" phenomenon may be observed.

First, the blood lead level drops during treatment. This is not a reason to interrupt therapy. Then, after treatment is stopped, the blood lead level almost invariably rises again. This phenomenon reflects a reequilibration of stored lead. The decision to repeat chelation therapy is based on the blood lead level after the "rebound."

Reduction of lead intake is urgent for all children in this category, both as part of immediate therapy and as part of the followup preventive procedure. Children receiving chelation therapy should not be released from the hospital until lead hazards in their homes and environment are controlled. Otherwise, suitable alternative housing must be arranged. Thus, the appropriate public agency in the community must be notified immediately so that environmental investigation and intervention can begin.

After their hospitalization and after lead has been removed from their environments, these children are still at high risk. Close followup, with blood lead and EP measurements, is required. At first, these tests should be done every 1 to 2 weeks. If the blood lead level rebounds to its pretreatment level, a repeat of the chelation therapy should be considered. If the blood lead level remains stable or shows a continual decline after the first few weeks, the interval between testing may be incrementally increased from 1 to 6

months until the blood lead and EP levels return to normal or the child reaches 6 years of age.

HIGH RISK

Many children in the high-risk category will have been given a calcium disodium EDTA mobilization test to determine whether chelation therapy is needed. If it is needed, inpatient chelation should be performed. Under some conditions, however, children without urgent risk factors may be treated as outpatients. Outpatient treatment should be reserved, however, for those centers capable of providing closely monitored outpatient care and followup supervision, and in those centers it should be provided only if the child's source of lead exposure has been eliminated (Piomelli et al., 1984). In addition, the parents should be cooperative and should demonstrate that they can follow instructions.

Followup of high-risk children should consist of blood lead or EP tests, or both, at least monthly (especially in the summer), until the sources of lead in their environments have been removed. If their blood lead or EP levels have declined or stabilized, the interval between testing may be incrementally increased, except in summer, from 1 to 6 months, until the blood lead and EP levels return to normal or the child reaches 6 years of age. Careful neurological and psychological assessment is advised so that any behavioral or neurological deviation can be detected early and proper therapy and school placement begun.

MODERATE RISK

Generally, children in this category do not require chelation therapy. Reducing lead intake from all sources and careful monitoring of the child usually suffices.

Until the lead hazards are eliminated from their environment, these children should be tested monthly in the summer and every 2 months in other seasons. If the blood lead and EP levels remain stable or show a continual decline after the first few months, the interval between testing may be incrementally increased from 2 to 6 months until the blood lead and EP levels return to normal or the child reaches 6 years of age.

NOTE: All children in the urgent-, high-, and moderate-risk categories may have concomitant nutritional deficiencies. These deficiencies may increase the child's risk from lead by increasing absorption, retention, and toxicity. All children in these categories should receive a careful nutritional evaluation, including appropriate laboratory tests. In addition to the care given for lead toxicity, nutritional therapy should be provided. When increased lead absorption is found, it may be particularly important to correct iron deficiency and maintain an adequate calcium intake.

LOW RISK

When tested, children in this category do not have significant evidence of lead toxicity. However, they require periodic screening until they reach their sixth birthday. Children whose elevated EP levels are not caused by

lead absorption should receive medical attention and care for the medical condition responsible for the elevation. Children with elevated blood lead levels but no evidence of toxicity should be evaluated monthly until lead toxicity can be ruled out. This can usually be done within 3 months.

In conclusion, the clinical management of children with lead poisoning must include appropriate treatment, adequate followup, environmental intervention, and family education. Chelation therapy is indicated for some children with lead toxicity. Using it indiscriminantly is unwise, but so is withholding or delaying it when it is indicated. The physician providing clinical management must know the current status of the child's environment. The optimal frequency of followup depends on many factors, including the child's age and environment and the trend in results of the child's tests.

VII. ENVIRONMENTAL EVALUATION AND LEAD HAZARD ABATEMENT

Environmental investigation and intervention should begin as soon as lead toxicity is confirmed. Lead hazards must be identified and removed from the environments of these children. Priorities for action should be determined by the child's risk classification. The higher the blood lead level and the lower the child's age, the higher the priority for removing the lead hazards. Children who require hospitalization and chelation therapy are at the highest risk of permanent neurologic damage from continued high-level exposure and

another episode of lead toxicity. Therefore, children in the urgent- and high-risk categories should receive first priority for environmental investigation and intervention. It is strongly recommended that abatement of lead hazards in a hospitalized child's home be completed during the first few days of the child's hospitalization.

Children in the moderate-risk category are next in priority. For them, identifying lead hazards and reducing lead intake are as much a medical necessity as clinical management. The effectiveness of environmental intervention is judged by the child's response and not by the services performed. Environmental management is not successful or complete until the child's EP and blood lead levels have declined and stabilized for at least 12 months. The identification and removal of one source of lead exposure does not necessarily mean that the child's exposure to lead has ended.

Because lead is a ubiquitous and powerful toxin, with no known beneficial function in the human body, the goal of prevention is to reduce children's exposure to lead to the maximum extent. Lead-based paint is the most common, remediable source of lead that causes symptomatic lead poisoning. Detailed procedures for removing lead paint from the home environment are described, but only general guidelines are given for controlling other sources of lead discussed in section III. Ideal prevention goals are given first; when these goals cannot be reached immediately, short-term, substitute goals are offered.

LEAD-BASED PAINT

The ultimate goal is to remove all leaded paint from housing in the United

States. Reaching that goal will be expensive. Short-term goals of partial removal help, but they tend to postpone efforts for complete removal.

All painted interior and exterior surfaces should be tested for lead. Portable x-ray fluorescence (XRF) analyzers are most convenient for identifying lead-based paint hazards. These instruments can measure lead content in paint surfaces within $\pm$ 0.2 mg/cm^2 of exposed surface. Readings of 0.7 mg/cm^2 are considered positive. The XRF analyzer is a probability sampling device, and reliability depends on repeated readings. If an XRF analyzer is not available, wet chemical methods of analysis must be used.

A lead-based paint hazard exists when (a) the XRF reading is positive and (b) the surfaces being tested are chewable or contain damaged (cracked, chipped, loosened, chewed) paint. Lead-based paint on intact walls, ceilings, or other surfaces that are not chewable does not constitute an immediate hazard. Inspectors should obtain measurements on any interior or exterior surface that may constitute a lead hazard. This includes walls, doors, window frames, baseboards, guardrails, fences, and sidings. Outside inspection should encompass garages and other adjacent structures as well as the main building.

Next, the inspector should classify each interior and exterior part of the building where lead is found according to the degree of hazard. If nonchewable surfaces with lead paint are smooth and intact and the supporting structure is sound, they do not present an immediate hazard and may be left

alone. Property owners and residents, however, should be warned that smooth surfaces containing lead can become hazardous if they are not properly maintained and are allowed to fall into disrepair. All lead-painted surfaces that are identified as positive by XRF (or wet chemical analysis) and that are in unsound condition are classified as immediate hazards requiring prompt abatement. This includes all wood trim--both interior and exterior--with blistering, scaling, peeling, or powdering paint and walls with unsound paint, painted plaster, or painted, peeling wallpaper. Floors and ceilings, if painted with lead-based paint and if in an unsound condition, are also included.

New information has revealed the importance of lead-bearing dust as another major hazard for young children. In the past, blistering, scaling, peeling, or powdering paint was frequently removed only to a level of 4 or 5 feet above the floor, because, usually, a small child can reach no higher. However, dust or paint chips from unsound lead paint above this level could fall into the child's play area. CDC now recommends that <u>all</u> unsound leaded paint be removed from the interiors of dwellings, including areas beyond the reach of children. Likewise, exterior leaded paint (on porches, woodwork, and walls) that either is in or can fall into the child's play area should be removed immediately. Places in and about the home where young children spend much of their time--namely, near windows, doors, and porches--are particularly hazardous.

In summary, paint in unsound condition or on chewable surfaces is classified as an immediate hazard requiring prompt abatement: other lead paint in sound condition may not require immediate attention, but it must be identified as a

potential hazard.

Next, some common methods for reducing lead-based paint hazards are outlined.

Phase I - Emergency Intervention

As soon as an elevated blood lead level is confirmed, residents should be advised to remove all scaling paint from places such as window sills, door frames, doors, and porch railings that are within easy reach of the child. A stiff brush should be used for this. Residents should also be advised to avoid inhaling the dust or contaminating other areas. The debris should be vacuumed and bagged for safe disposal. Then the area should be thoroughly scrubbed, preferably with high-phosphate detergents such as Spic and Span (Milar and Mushak, 1982). If a crib is next to a surface with scaling paint, the crib should be moved away. Similarly, a piece of furniture should be moved to prevent the child from reaching areas of scaling paint. In the past, it was advised that window sills and other wood trim with peeling paint be covered with masking tape or some other adhesive-backed paper. This is no longer recommended. Inquisitive young children often remove this tape, thereby rendering the technique ineffective. Families should be instructed on ways to keep these areas free from loose or flaking paint until more definitive steps can be taken to reduce the hazard. Housekeeping techniques such as frequent wet mopping and damp dusting are essential in maintaining a reduced level of hazard.

Phase II - Long-Term Hazard Reduction

Only when an old dwelling with lead-based paints is gutted and completely restored can the lead hazards be considered "permanently abated." Less extensive, commonly used procedures may be called "long term"; however, how long the hazard will remain under control depends on such factors as the thoroughness of the procedure, the soundness of the underlying structure, and the condition of the plumbing. Increased moisture from leaky pipes behind walls can quickly cause paint that was smooth and intact to blister and scale.

Abatement entails four steps:

1. Removing lead paint from wood trim or walls.
2. Thorough vacuuming to clean up the debris.
3. Wet scrubbing for maximum elimination of fine lead-bearing particles.
4. Repainting the area with lead-free paint (that is, paint containing less than 0.06% of lead in the final dried solid).

The property owner's responsibility is not met until all four steps have been completed.

Just prior to and during abatement, certain precautions are essential. Carpets, rugs, upholstered furniture, bedding, clothing, and eating and cooking utensils must be sealed as tightly as possible in plastic to protect them from the enormous increase in lead-bearing dust created by the removal procedures. Once items such as rugs are impregnated with fine, lead-bearing particles, it is almost impossible to remove the lead (Milar and Mushak,

1982). When feasible, this work should be carried out in one room at a time, with the room closed off and all furnishings removed. Until steps 1, 2, and 3 of the cleanup process are completed, all young children and pregnant women should live elsewhere both day and night. If this is not possible, they, as well as the child with the index case, should have serial blood lead tests before, during, and after the abatement work. Those doing the work should comply with OSHA standards; they should use respirators and wear coveralls, which must not be taken to the workers' homes for laundering.

Walls

Removing lead paint from walls, particularly lead paint applied to plaster, is usually difficult. In most cases, a barrier, such as wallboard, hardboard, fiberglass, plywood paneling or a similar durable, fire-resistant material, can be placed over the lead paint on the walls. These materials must be firmly nailed, cemented, or glued in place to prevent the child from removing them. The barriers should be verminproof, and in certain areas of the dwelling (that is, next to furnaces and stoves and in common hallways), fire retardant. Wallpaper painted with lead paint should be stripped off to the maximum extent possible.

Woodwork

Lead-based paint in unsound condition on both interior and exterior wood trim (for example, window units, door units, stair risers, bannisters, and railings) presents considerable danger for children. Paint can be removed from wood surfaces by heat (from gas torches and heat guns), sanding,

scraping, and with liquid paint removers. All of these methods are hazardous. Most solvents in liquid paint removers evaporate rapidly and are flammable and toxic. These removers must be used with the utmost caution and only in well-ventilated areas with proper protective clothing and equipment. When the underlying wood has rotted, no attempt should be made to remove the paint. Instead, the wood should be replaced, including, when necessary, entire window units and doors or door frames. Exterior rotted wood should also be replaced. When torches, heat guns, and sanding devices are used to remove paint, air lead levels increase enormously in the work area. Of these, sanding is by far the worst offender. It also produces the greatest deposition of lead in dust, with rates as high as 10 mg of lead/sq ft/hour (Inskip and Attenbury, 1983). Therefore, fine sanding down to the bare wood surface is not recommended. Scraping the surface after a heat gun has been used will probably produce fewer fine particles than sanding.

The above information emphasizes the urgency of proper cleanup after lead-based paint has been removed. After the dust has settled, the entire area, including walls, floors, and ceiling, should be vacuumed, preferably with an industrial vacuum cleaner. All surfaces should be wet-scrubbed with phosphate-containing detergents. Immediately thereafter, all surfaces from which paint has been removed should be repainted with lead-free paint. For safe disposal the debris should be placed in a toxic waste dump approved by the Environmental Protection Agency, not in an ordinary landfill or storm or sanitary sewer system. For best results, the wet cleaning procedures should be repeated (Milar and Mushak, 1982). Workers who remove the paint should be responsible for the cleanup, inasmuch as many of the affected families have neither the equipment nor the resources to carry out an adequate cleanup.

Supplemental Addresses

Children often spend substantial amounts of time with relatives or babysitters who live at a different address. If lead-based paint in unsound condition is found at these addresses, it should be removed in the manner described. Similarly, day-care centers and other facilities may be located in old buildings with lead-based paint. These, too, should be checked and handled accordingly.

Followup

The effectiveness of the initial abatement can be determined only through coordinated medical and environmental followup. When the initial abatement has been inadequate, a high recurrence rate of blood lead levels above 50 ug/dl has been found (Chisolm, 1983). Ideally, a communitywide code-enforcement program should be developed to remove all lead-based paint in housing. But, until then, the appropriate governmental unit in which the child lives is responsible for identifying and abating lead hazards for children with lead toxicity. Removing lead hazards in housing is the major factor in the success of a lead poisoning prevention program.

AIRBORNE LEAD

Blood lead levels are decreasing as the use of leaded gasoline decreases (Annest et al., 1983). In terms of reducing background blood lead levels, removing lead from gasoline as rapidly as feasible is probably the most important public health measure.

Emissions from industrial sources should be reduced sufficiently to achieve the current ambient air lead standard. New factories, as part of their licensing specifications, should be required to have minimal lead emissions.

The public should be informed about the hazards associated with burning old battery casings, colored newsprint, waste oil, and lead-painted wood.

SOIL AND DUST

The optimal goal is to prevent lead from being transferred from any source to soil and dust. For the goal to be reached, air lead levels must be reduced to near zero. For those areas where concentrations of lead in soil and dust are high, large-scale excavation of soil or relocation of populations is the ideal means of reducing the exposure of children to lead.

When the lead content of household dust is high, wet-mopping and other cleanup measures help reduce children's blood lead levels (Charney et al., 1983). These measures provide a reasonable, short-term and mid-term solution to the problem of contaminated house dust.

In severely contaminated residential areas, unless an effective barrier can be established between the children and the soil, surface soil must be removed and replaced with soil having a low lead content.

FOOD AND WATER

The lead content of air and soil, important contributors to the contamination of food and water, should be reduced. Food cans should be made so that lead does not leach from soldered seams. Lead is also added inadvertently to foods during processing and handling (Wolnik et al., 1983). Although the percentage of canned foods packaged in cans with lead-soldered side seams has declined substantially, some are still packaged this way. These foods should not be stored in the opened cans because, after the cans have been opened, even more lead migrates from the side seam into the food.

When feasible, lead plumbing and lead water mains should be replaced. Water from taps in the home should be assessed for lead content. If a hazard is found, consumers should be educated to run water for several minutes before drinking it and not to drink water from the "hot" side of the tap. Acidic water supplies should be alkalinized to help prevent leaching.

OCCUPATIONAL

Ideally, engineering features should prevent workers from being exposed to lead dust and vapors. When workers are exposed, compliance with Occupational Safety and Health Administration (OSHA) regulations appears to be effective in protecting them and in preventing them from transporting lead home to children. Under the OSHA lead standard, factories that use lead must provide workers with facilities for showering and changing clothes and shoes before going home from work. This standard now applies only to industries covered by OSHA regulations. For the protection of children, it should be extended to all industries that use lead. The prevention of lead exposure to the fetus needs special emphasis. Women of childbearing age should be excluded from working at jobs where significant lead exposure occurs.

LEAD-GLAZED POTTERY

All glazed pottery used for foodstuff should be free of leachable lead. Hobbyists and consumers should be educated to the risks associated with pottery glazes. Consumers should not use pottery for cooking or for storing food or beverages unless the pottery has recently been determined to be free of leachable lead.

VIII. HEALTH EDUCATION

The community and especially parents of preschool children who live in older, deteriorating neighborhoods should be informed at every available opportunity

of the need to have children screened periodically for lead poisoning. Basic preventive measures should be emphasized. These include frequent wet mopping and vacuuming of accessible paint flakes and dust to reduce potential lead hazards in the child's environment. The danger of ingesting paint chips, dust, and soil should be stressed. Older siblings of children at high risk should also be informed about the sources and risks of lead poisoning because they often take care of younger children.

If a child is screened and the lead level is not elevated, the risk remains, and until the sixth birthday, rescreening is required, particularly during the summer. Until hazard-free housing is available for all and other high-risk sources of lead are removed, periodic screening will reduce the risk of lead poisoning.

Education should start when the child is screened, and physicians, nurses, environmentalists, and aides should reinforce it at every opportunity. When a child is found to have lead toxicity, education of the family is essential for successful followup of the child. The family must be fully informed about the condition and the clinical and environmental actions to follow. Health professionals must emphasize the importance of the family's understanding the child's condition, its cause, and the possible result of lead toxicity. In addition, they should stress the importance of the child's having a balanced diet that includes enough calcium and iron.

IX. REPORTING LEAD TOXICITY AND ELEVATED BLOOD LEAD LEVELS

Primary care physicians and persons in charge of screening programs should report both presumptive and confirmed cases of lead toxicity to the appropriate health agency, and laboratories performing blood lead or EP tests should report any abnormal results to the appropriate health agency.

Use of trade names is for identification only and does not constitute endorsement by the Public Health Service or by the U.S. Department of Health and Human Services.

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